AF was shown in 78% (14/18): APC only initiated AF, not involved in the maintenance of AF. Triggering AF by APC was related to wave break-up at the septopulmonary bundle near the RSPV ostium in 55% (10/20), posterior roof of LA in 35% (7/20). 2) Focally driven AF was shown in 22% (4/18): APCs not only initiated AF but also played a role in the maintenance of AF. During AF, these APCs were continuously generated from the arrhythmogenic foci and activated the LA. The wave propagation patterns of induced AF were different from those of spontaneous AF in 54.5% of patients. APC originated from single PV was in 45% (9/20), two PVs 15% (3/20) and more than three PVs 20% (4/20). Non-PV foci were identified in 37.5% (6/16) of the patients and mostly located at the posterior roof of the LA (12.5%, 2/16).

Conclusion: A non-contact mapping system may facilitate identification of the mechanisms and the relationship with the underlying anatomical structures responsible for the initiation and maintenance of AF.

1089-14 C-Reactive Protein in Atrial Fibrillation: Evidence for the Presence of Inflammation in the Genesis and Perpetuation of the Arrhythmia

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Background: Atrial fibrillation (AF) is associated to a number of clinical entities with an underlying acute or chronic inflammatory process. It is possible that inflammation could contribute to the initiation and perpetuation of this arrhythmia.

Aims: To evaluate the presence of an inflammatory process in patients with newly diagnosed non valvular AF.

Methods: We studied 109 consecutive patients admitted for newly diagnosed AF. Forty patients resulted in chronic AF and 69 had paroxysmal AF. All patients had determinations of C reactive protein (CRP) at baseline and at 30 days, 68 had CRP at 1 year of follow-up. In addition the patients were evaluated clinically and with general laboratory tests. A transesophageal echocardiogram (TEE) was performed within the first 24 hours of admission to evaluate left atrial and ventricular dimensions, LV function and the presence of spontaneous echo contrast or intracavitary thrombus.

Results: Mean age was 67 ± 14 years and 50% were >70 years. Hypertension was found in 57%, diabetes in 17% and cardiomyopathy in 36% of cases. A history of previous systemic embolism was present in 6 patients. Relevant TEE findings were left atrial diameter >45 mm in 52%, left ventricular dysfunction in 15% and the presence of spontaneous echo contrast and or LA thrombus in 53% of cases. CRP levels in the whole group were 1.0±1.8 mg/dl, 0.9±1.4 mg/dl in patients with paroxysmal AF and 1.1± 2.4 mg/dl in patients with chronic AF (p=0.001 AF group, p=0.01 paroxysmal AF group and p=0.01 chronic AF group vs 20 normal subjects in sinus rhythm). CRP levels correlated with other laboratory markers of inflammation (VHS, p=0.003 and WBC, p= 0.004) and with the presence of LV dysfunction in TEE (p = 0.02) in multivariate analysis. At 30 days there was a significant decrease in CRP both in chronic and paroxysmal AF (p= 0.048 and 0.03 respectively). At 1 year of follow up mean CRP levels in patients that were still in AF were 1.2±1.8 mg/dL, whereas in those with sinus rhythm were 0.5±0.5 mg/dL, p = 0.048)

Conclusion: This study provides evidence for the presence of an inflammatory process in patients with newly diagnosed non valvular AF. The persistence of inflammation is associated with chronic AF at 1 year of follow up.

1089-15 Plasma Amino-Terminal Pro-B-Type Natriuretic Peptide Predicts Postcardioversion Reversion to Atrial Fibrillation

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Background: With mounting evidence that rate control plus anticoagulation is the treatment of choice for chronic atrial fibrillation (AF), there is a need to improve patient selection for electrical cardioversion (CV) from AF by more accurate prediction of those likely to exhibit sustained return to sinus rhythm.

Methods: Plasma cardiac natriuretic peptides (NPs, including ANP, BNP and pro-amino-terminal BNP [N-BNP]) and adrenomedullin (ADM) were assayed before, immediately after and six weeks after cardioversion in 151 patients with chronic AF.

Results: Plasma NPs (but not ADM) fell significantly immediately postsuccessful CV (all p<0.0001) but not in 15 patients in which CV failed. At six months postCV, 65 patients remained in sinus rhythm whilst 86 were in AF. Plasma NPs (but not ADM) were significantly higher at all three sampling times in those destined to be in AF at six months. Kaplan-Meier analysis indicated significant separation of AF-free survival curves by median preCV and immediate postCV levels of BNP (p=0.039 and 0.0024 respectively) and N-BNP (p=0.022 and 0.010) but not ANP (p=0.075 and 0.063) or ADM (p=0.425 and 0.253). Tertiles of precardioversion N-BNP differed significantly for risk of AF at six months (39, 60 and 70% risk in first, second and third tertiles respectively; p=0.006). Multiple regression analysis indicated both pre and immediate postcardioversion levels of BNP (p=0.01 and 0.009) and N-BNP (p=0.006 and 0.011) were predictive of reversion to AF independent of age, left ventricular function or the prophylactic prescription of beta blockers/amiodarone.

Conclusions: In patients with chronic AF, those with plasma N-BNP concentrations elevated above the lowest tertile may be more rationally treated with rate control and anticoagulation rather than undergoing single or serial electrical cardioversion.

1089-16 C-Reactive Protein and Microalbuminuria Are Associated With Atrial Fibrillation

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Background: Atrial fibrillation (AF) is a major risk factor for stroke and cardiac events. Early identification of subjects at risk for AF might help to prevent the onset of AF. The inflammatory marker C-reactive protein (CRP) and microalbuminuria are easy to obtain risk indicators for ischemic heart disease. Ischemic heart disease is one of the most important predisposing factor for AF. In this study, we hypothesized that CRP and microalbuminuria are associated with AF.

Methods: In a large population based survey (8501 subjects), atrial fibrillation was measured by one 12 lead electrocardiogram and defined according to Minnesota codes 8.3.1 and 8.3.3. Mean age was 49 ± 12.7 years and 49.8% male. Microalbuminuria was defined as 30-300 mg/24h. High sensitive CRP was dichotomized (three lowest quartile, CRP< 2.97 mg/l versus the highest quartile, CRP> 2.97 mg/l). The following cardiovascular risk factors were assessed and used in the analysis: age, sex, hypertension, hypercholesterolemia, obesity, diabetes mellitus, smoking, ischemic heart disease, and left ventricular hypertrophy.

Results: Atrial fibrillation was present in 75 (0.9 %) subjects. AF was significantly associated with the highest quartile of CRP (2.63 [1.71-4.05], p<0.001) and microalbuminuria (3.46 [2.18-5.49], p<0.001). When all cardiovascular risk factors were entered simultaneously, CRP (2.09 [1.29-3.40], p=0.003) and microalbuminuria (2.06 [1.21-3.50], p=0.008) remained significantly associated with AF. The combination of the highest quartile of CRP and the presence of microalbuminuria showed a higher association with AF (7.36 [4.16-13.01], p<0.001), even after adjusting for all cardiovascular risk factors (4.07 [2.08-7.95], p<0.001).

Conclusion: In this study, we demonstrated that C-reactive protein, microalbuminuria, and the combination of both are associated with atrium fibrillation independent of cardiovascular risk factors.

1089-17 Is Atrial Fibrillation an Inflammatory Disease Reflected by Elevated C-Reactive Protein?

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Background: Inflammation is implicated in the pathogenesis of cardiovascular diseases. C-reactive protein (CRP), a marker of systemic inflammation, predicts increased risk of coronary events and stroke. Atrial fibrillation (AF) is associated with cardiac structural changes, including atrial fibrosis, which may have an inflammatory basis. In a recent small retrospective study, patients (pts) with AF had higher CRP levels than controls. We prospectively tested the hypothesis that CRP is a risk factor for AF in a large cohort.

Methods: Consecutive pts (N=3,357) free from myocardial infarction had a CRP level drawn at the time of angiography. These pts were enrolled in the Intermountain Heart Collaborative Study between 1994-2001. Those with ≥1 ECG showing AF during the study period formed the disease group (n=347). Those with neither ECG nor clinical (ICD-9) evidence for AF comprised the control (C) group (n=2449). The other 561 were excluded. CRP was measured by fluorescence polarization immunoassay (Abbott). Logistic regression assessed quartile (Q) of CRP and 13 other clinical and angiographic variables as predictors of AF.

Results: Average age was 63±12 years; 33% were women; 61% had significant coronary artery disease (CAD), and 26%, no CAD, median CRP was 1.21 mg/dL. AF pts were older (by 7 years) and more frequently had a history of heart failure (HF) than C pts (41% vs. 10%) (both p<0.001). CRP was higher in AF than C pts (1.97 vs. 1.51 mg/dL, p<0.001). Q-CRP was a univariable predictor of AF (odds ratio [OR]=1.39/Q, CI 1.25-1.55, p<0.001). After adjusting for age and HF, the predictive value of Q-CRP was reduced but retained significance (OR=1.20/Q, CI 1.07-1.34, p=0.002). Further adjusting for 11 other variables had little effect (OR=1.19/Q, CI=1.06-1.33, p=0.003)

Conclusion: Increasing CRP independently predicted an increased risk of AF among a large, prospectively studied patient cohort assessed angiographically. These results support the proposal that AF pathophysiology includes an inflammatory mechanism, that elevated CRP is a new risk marker for AF propensity, and that AF therapies targeting inflammation are worthy of testing.

1089-18 Altered Collagen Subpopulations and the Matrix Metalloproteinase Regulation in the Development of Atrial Fibrillation in Atrial Dysfunction

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Background: Myocardial remodeling has been found not only in ventricle, but also in atrium in dilated cardiomyopathy and heart failure. We hypothesize that atrial extracellular matrix remodeling may be responsible for the AFib recurrence and maintenance in atrial dysfunction.

Methods and Results: Tissue samples from 55 hearts explanted from patients with dilated cardiomyopathy and end-stage heart failure underwent heart transplantation were examined. Nineteen patients had permanent AFib (PmAF), 20 had persistent AFib (PsAF) and 16 had no documented AFib (NAF). Sixteen donor left atrium were used as normal control (CN). Fibrillar collagen subtype I and III distributions in the left atrium (LA) were examined by immunofluoresent staining. LA metalloproteinase-1 and 2 (MMP-1, MMP-2), tissue inhibitor of metalloproteinase-1 and 2 (TIMP-1, TIMP-2) proteins were evaluated by western blot. Type I collagen volume fraction (CVF-I) increased significantly